organisms must sometimes choose between allocating their limited resources to somatic maintenance or to reproduction (reviewed in [17]). For example, such a shift might underlie the downregulation of some metabolic genes in *D. melanogaster* females after mating [2]. Increased reproduction can come at a cost to an individual's survival; on the other hand, relatively high investment in the soma can result in a cost to reproduction. Previous studies in insect reproduction offer several precedents for the idea that males might help mitigate such tradeoffs in their mates. Males may provide females with nuptial gifts, for example of secretions from male glands (reviewed in [5]), of specific compounds such as salt [18], or elements such as phosphorus [19] that can nourish the female and assist with egg provisioning. Carvalho *et al.*'s [1] results suggest a new way in which a *Drosophila* male can influence resource input into his mate: he simply induces her to eat! Increased feeding may in turn allow a female to put relatively more investment into egg production, or to increase resources available for reproduction without changing relative resource allocation patterns.

Finally, the results presented here may merit consideration in practical applications, for example in the control of some disease vectors. Food plays a very important role in mosquito reproduction: blood meals are nearly always essential for the female mosquito to produce eggs, and some studies (reviewed in [20]) have shown that mating status can impact a female mosquito's behavior and physiology. Given that a male-derived peptide can change the feeding behavior of *D. melanogaster* (a Dipteran, as are mosquitoes), might a similar phenomenon operate in any mosquito? If so, it might help us to understand and possibly control the transmission of some vector-borne diseases.

Carvalho *et al.*'s [1] results leave us hungry for more. Mating-induced eating represents a novel post-copulatory behavior, which to our knowledge has not been demonstrated in any other species. The topic is made all the more 'appetizing' by its broad range of implications: to regulation of post-mating behaviors, life-history trade-offs and, possibly, for practical applications.

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Department of Molecular Biology and Genetics, Cornell University, Ithaca, New York 14853, USA.
E-mail: mfw5@cornell.edu

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Myopia: The Importance of Seeing Fine Detail

Eye growth and myopia development are controlled by the retina. What properties of the image tell the retina how the eye should grow? A recent study has shown that, in chickens, fine details are necessary to prevent the development of myopia. Should we carefully avoid any defocus to avoid becoming myopic?

Frank Schaeffel

The organ with the highest geometrical precision in the body is most likely the eye: for a human to be normal-sighted (or emmetropic), which means being able to see sharply at far distances, the geometrical length of the eye must be matched to its optical focal length with a precision of about 0.2 percent, less than the thickness of an eyelash. An increase in eye length of just 0.1 millimetre is sufficient to cause a measurable decline in visual acuity for distant objects — myopia. When this happens, the sharpest image projected by cornea and lens is formed, not on the photoreceptor layer of the retina, but in front of this layer. Myopia is quite frequent

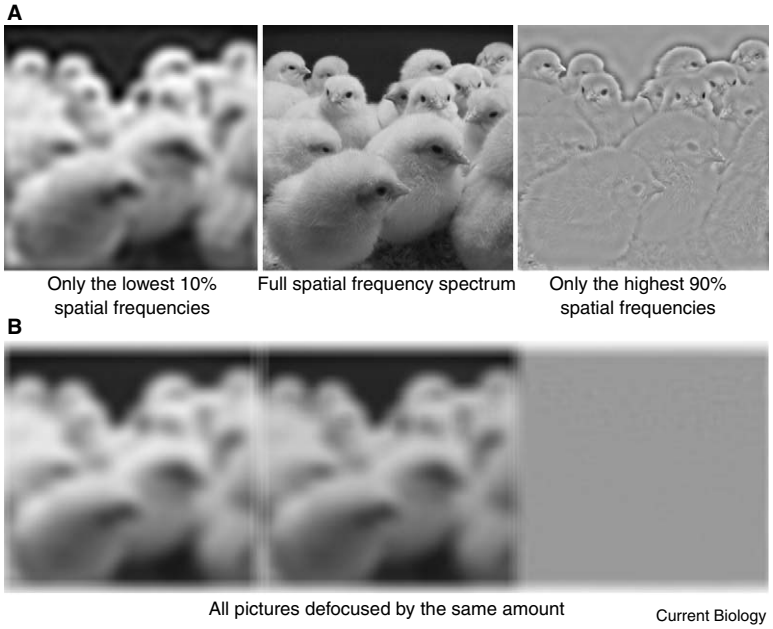

Figure 1. The concept of low and high pass filtering and the effect of defocus.

(A) A view of little chickens in their cage: middle, the full spatial frequency spectrum provided by the camera; left, low-pass filtered image; right, high-pass filtered image. Using the software NIH image (publicly available at http://rsb.info.nih.gov/nih-image/), either the lowest 10% of the spatial frequencies were included (left) or the lowest 10% were cut out (right). (B) When the pictures from (A) are defocused, there is little change in the low-pass filtered image (left) but all information is lost in the high pass-filtered image (right). A mechanism that controls emmetropization based on only high spatial frequencies would have a narrow dioptric operation range.

in the industrial nations: about 30 percent of the young adults are affected, and there is evidence that this proportion is increasing [1].

Recent epidemiological studies (reviewed in [1]) suggest that environmental factors must be responsible for the increase in myopia, most likely heavy 'near work', such as extensive reading or computer work with short viewing distances. But what exactly stimulates the eye to grow longer during 'near work'? This question is fundamental to the understanding of myopia and has not yet been solved. As they report in this issue of *Current Biology*, Hess *et al.* [2] have now shown that fine details must be present in the retinal image to prevent the development of myopia — at least in little chicks that were raised with artificially generated, well-defined viewing targets in front of their eyes.

About 30 years ago, it was shown that a clear image on the retina is necessary to prevent exaggerated eye growth in young chickens. If the image is blurry, for example because the eyes are covered with diffusing goggles, enormous amounts of myopia develop in only a few days, producing up to 20 diopters myopia, with eyes that are 20 percent longer than normal [3]. This has been called 'deprivation myopia', because it results from deprivation of a clear retinal image. Later, it was shown that deprivation myopia can be induced in most vertebrates, including primates; it can also occur in children with optical conditions that degrade the image on the retina, cataracts for example. It is tempting to speculate that human myopia results generally from problems with retinal image quality, perhaps as a result of inaccurate accommodation during reading, aberrant optics or other factors.

But what exactly are the properties of the retinal image that are so deleterious for eye development? If this were known, one could try to avoid these conditions and reduce the risk of developing myopia. A few variables have previously been tested in chickens: these studies found that the more frosted the diffusers over the eye [4], or the lower the contrast in the retinal image [5] the worse the myopia that develops. Myopia was also induced if chickens wore 'sun glasses', dark filters that reduced the brightness of the retinal image [6]. But it remained unclear precisely which image property is responsible for the development of myopia.

Hess *et al.* [2] now report an elegant study which has further defined the properties of the retinal image that control deprivation myopia. To understand their study, it is necessary to understand the concept of Fourier analysis. At the beginning of the 19th century, Jean Baptiste Joseph Fourier showed that any function can be represented as a superposition of sine waves with different frequencies, amplitudes and phases; he also provided equations to calculate these sine waves. As an image can be considered as a two-dimensional function of brightness versus position, it can also be decomposed into Fourier components. The underlying sine wave components are called 'spatial frequencies' and their units are either number of waves per visual angle (cycles per degree) or per linear distance (cycles per millimetre). A nice feature of Fourier decomposition is that the images can now be filtered: low or high spatial frequencies can be left out, and one obtains images that look either blurry or sharper than normal, respectively (Figure 1A).

Hess *et al.* [2] forced young chickens to view, with one eye, patterns that had either more low or more high spatial frequencies than normal; they assumed that, if the respective retina had enough information to guide eye growth, then the eye would not develop differently from its fellow allowed to experience normal vision. They found that high spatial frequencies were necessary to 'keep the eye on track'; if only low spatial frequencies were presented, the eyes developed deprivation myopia. Furthermore, they found that the phases between the sine wave components were not important. Pattern vision requires that the phases are aligned, and scrambling the phases produces images that look

meaningless — but these patterns were sufficient to produce normal eye growth (this means that the common expression 'form deprivation myopia' is misleading, as 'form vision' appears to play no role). For the control of eye growth, therefore, the retina does not seem to care about object or form vision — it is only the high spatial frequency content that is important.

What does this mean for human myopia development? If high spatial frequencies in the retinal image are required to avoid myopia, even small amounts of defocus could be a major risk factor. This is illustrated in Figure 1: all three images in Figure 1A — a normal image in the middle, flanked by a low-pass-filtered image on the left and a high-pass-filtered image on the right — are defocused in Figure 1B. The image on the left is not much affected by the defocus, but in the image on the right, the high spatial frequency components are lost as a result of the defocus. If the high spatial frequency components are so important, defocus would become a rather critical experience. It would not be advisable to under-correct myopic people, as this could stimulate deprivation myopia. In fact, a widely cited recent study [7], comparing myopia progression in fully-corrected and undercorrected children, claimed that undercorrection accelerates myopia progression.

But this cannot be the whole story. A control system for eye growth that is dependent only on high spatial frequencies would seem inherently unstable. The signal would be lost with small amounts of defocus, and with increasing myopia, there would be further loss of high spatial frequencies, accelerating myopia in a positive, feed-forward fashion. It is hard to believe that nature would rely on an open loop system to control such an important variable like eye growth.

In addition to the retinal mechanism that determines the spatial frequency content and 'prevents deprivation myopia', there is another mechanism that determines whether the focal plane is in front or behind the photoreceptor layer — it 'measures' the vergence of rays. This mechanism provides a strong inhibitory signal for eye growth if the image is in front of the retina, even though high spatial frequency components are lacking ([8], reviewed with additional data in [9]). The inhibitory signal is much more powerful than the one that causes deprivation myopia. For example, four periods of only two minutes a day with positive lenses block deprivation myopia completely, even though the eye was covered with frosted goggles for all of the rest of the day [10].

So there is still much to be learned about the visual control of eye growth. One big question is: what could be the biological sense of a retinal mechanism that produces myopia by default when the high spatial frequencies are lacking from the retinal image — the *open loop condition*, as studied by Hess *et al.* [2]? In particular, why is this mechanism needed when there is an additional *closed loop feedback system* for the control of axial eye growth that uses the *sign* of the optical defocus as an error signal [9]? And how could the retina determine the sign of imposed defocus, which occurs 'in a matter of minutes' as shown by Zhu *et al.* [11]. Detection of the sign of defocus would appear to be a more demanding task than 'measuring' the spatial frequency content of an image; but measuring spatial frequencies may not be enough for the development of normal vision.

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Section of Neurobiology of the Eye, University Eye Hospital, Calwerstr. 7/1, 72076 Tuebingen, Germany.
E-mail: frank.schaeffel@uni-tuebingen.de

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X-Inactivation: Close Encounters of the X Kind

X chromosome inactivation ensures equal dosage of X-linked genes between male and female mammals. Two new studies have shown that the initiation of inactivation is preceded by X chromosome pairing; their results implicate this pairing in the choice and counting functions of X chromosome inactivation.

James M.A. Turner

In mammals, males have one X chromosome (XY) and females two (XX). In order to ensure that the levels of X-gene products are equal between the two sexes, one X chromosome in each female cell is inactivated early in embryonic development. X chromosome inactivation (XCI) has three broadly defined steps [1]. In the first step, 'counting', the cell must register that more than one X chromosome